Parasitic Causes of Pericarditis

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ALTHOUGH MYOCARDITIS is frequent in African trypanosomiasis and American trypanosomiasis (Chagas' disease), involvement of the heart is an uncommon association with other parasitic diseases and pericarditis is even more rare.

Pericardial effusions containing larvae have been described in severe infections with Trichinella spiralis, but the myocarditis in these patients overshadows the pericardial involvement. Toxoplasmosis has been reported as a cause of pericarditis.[67,68] Usually, the diagnosis has been made by serologic methods. One patient with a serologic response diagnostic of toxoplasmosis had pericarditis with progressive pericardial constriction.[69]

Microfilariae have been found in the fluid aspirated from a pericardial effusion,[70] and constrictive pericarditis has been described in a patient in whom a pericardial effusion associated with filariasis developed.[71]

Pericarditis is an uncommon but well described complication of amebic liver abscess. In almost all patients, the abscess has been located in the left lobe of the liver, but there have been rare instances in which abscesses of the right lobe have extended into the pericardial sac.[72]

Initially, the pericardial involvement may be a serous or serosanguinous effusion (presuppurative stage), but this progresses to a purulent effusion when there is direct communication between the liver abscess and the pericardial cavity (suppurative stage). Although many patients will have a history of travel in areas of the world where amebiasis is common, it should be emphasized that amebic liver abscess and pericarditis occur sporadically in temperate areas, including the United States. The clinical manifestations usually begin with signs and symptoms of a left lobe liver abscess. Epigastric or low retrosternal pain, nausea, vomiting, left shoulder pain, cough, dyspnea, low grade fever, weight loss and tender hepatomegaly are common findings.[73,74] A few patients present solely with symptoms of pericarditis. Pericardial involvement is frequently suggested by rapid progression of symptoms and the appearance of signs of pericardial tamponade. Enlargement of the cardiac outline and the "water bottle" configuration of pericardial effusion are commonly seen on chest roentgenograms. Electrocardiography shows changes seen in other forms of pericarditis. Low voltage is also frequently present.

Patients with amebic pericarditis who are misdiagnosed or receive inadequate treatment usually die of pericardial tamponade. When adequate therapy is initiated early, two thirds of the patients may be expected to survive.[74] A few patients have developed constrictive pericarditis following resolution of the acute problem and in spite of adequate antiamebic therapy.

Diagnosis of amebic pericarditis is dependent on detecting the underlying liver abscess by hepatic scan or echogram. Results of serologic tests for amebiasis have been positive in more than 90 percent of patients with hepatic amebiasis. Examination of material aspirated from the liver or pericardium may reveal trophozoites of Entamoeba histolytica. Fecal examination should be carried out but findings are usually negative for amebae.

Immediate treatment is directed toward relieving or preventing pericardial tamponade. This may be done by percutaneous needle aspiration, repeated as frequently as necessary,[74] or by the creation of a subxiphoid pericardial window under local anesthesia.[75] Specific antiamebic therapy should be instituted immediately. Metronidazole has become the drug of choice for the treatment of hepatic amebiasis; however, doubt has been raised about its efficacy in patients with pericardial involvement.[72] Metronidazole attains a high concentration in the liver, but not in the myocardium. Until more information is available, treatment of amebic pericarditis should consist of a combination of chloroquine phosphate 250 mg four times a day for two days then twice a day for 28 days *and* emetine hydrochloride 1 mg per kg of body weight with 65 mg maximum given intramuscularly daily for 10 days (alternate dihydroemetine dihydrochloride 1 to 1.5 mg per kg of body weight for ten days; maximum total dose 1 gram). A luminal amebicide such as diiodohydroxyquin 650 mg three times a day for 21 days will reduce the likelihood of a persistent, undetectable enteric infection with E histolytica.

Patients should be followed closely after treatment for signs of constrictive pericarditis.

Hydatid disease is caused by the ingestion of

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eggs of the tapeworm Echinococcus granulosus. The source of the eggs is usually dog feces, but other carnivores are capable of harboring the adult tapeworm. The ingested eggs hatch in the intestine, and the liberated embryos burrow through the intestinal mucosa and enter the vasculature. They are carried to various tissues where they develop into the larval form, the hydatid cyst. The embryos must pass the filters of the liver and lungs before reaching the blood supply of the myocardium. Therefore, it is not surprising that the majority of patients with hydatid disease have cysts localized to the liver or lungs. Myocardial involvement is seen in only 0.3 to 3.3 percent of cases of hydatid disease.[76]

Pericardial involvement occurs when the cyst in the myocardium ruptures into the pericardial sac. This event frequently is not clinically detectable, or it may lead to a fibrinous or fibrinopurulent pericarditis.[76] In addition, larval tissue may produce secondary cysts. As the secondary cysts enlarge, they may cause a pericardial reaction and the signs and symptoms of constrictive pericarditis.[77] An acute pericarditis has been reported in association with allergic manifestations including anaphylaxis.[78,79] This condition may progress to massive pericardial[80] effusion with tamponade. A fibrotic reaction of the epicardium which impaired cardiac function and mimicked constrictive pericarditis has also been reported.[80]

The diagnosis of echinococcosis may be suspected in patients who have lived in endemic areas. There may be roentgenographic features of unusual bulges in the cardiac silhouette, intracardiac or pericardial calcifications and, infrequently, the finding of hydatid cysts in the liver or lung. The electrocardiogram may show diminished R waves or the presence of Q waves over the area of the intracardiac cyst. Deep T wave inversion may also be seen in the affected precordial leads.

Skin testing with hydatid fluid (Casoni test) may show reactivity, but false negatives are not uncommon. Serologic tests may be helpful but a negative result does not rule out hydatid disease.

Surgical removal of the cysts is the only effective treatment. Myocardial cysts may be technically very difficult to excise depending upon their location. It is essential that remnants of larval tissue be destroyed or secondary cysts may develop. Formalin, alcohol and 33 percent sodium chloride solution have been used to sterilize cyst cavities. Because of the nature of the myocardium, the concentrated salt solution is considered the agent of choice.[77] The use of formalin in the pericardial sac may cause a chemical constrictive pericarditis.

Patients who have had pericardial or myocardial cysts removed require careful follow-up. Electrocardiographic changes may indicate the development of secondary cysts and the need for reoperation.

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